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USING ELECTRONIC MEDICAL RECORDS (EMRS) TO ASSESS ANTICOAGULATION STATUS ACCORDING TO STROKE RISK IN PATIENTS WITH ATRIAL FIBRILLATION IN AMBULATORY CARE SETTINGS

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OBJECTIVES: To assess the prevalence and adequacy of warfarin use in patients with atrial fibrillation (AF) according to stroke risk, as assessed by $CHADS_2$ scores, in a nationally representative patient sample. **METHODS:** The data source was GE's Medical Quality Improvement Consortium (MQIC) database (February 2009) containing electronic medical records (EMRs) data on >11 million patients in the U.S. Eligible patients were those with a diagnosis of AF, ≥age 40 years at AF diagnosis, and with no use of warfarin or antiplatelet agents at anytime prior to AF diagnosis. Ineligible patients were those who might have other reasons, such as cancer or orthopedic surgery, to be hypercoagulable (i.e. other justifications for different anticoagulant use or INR ranges), or those already on antiplatelet agents or warfarin prior to AF diagnosis. $CHADS_2$ stroke risk scores were assigned on the basis of conditions prior to or coincident with the AF diagnosis (congestive heart failure, hypertension, diabetes mellitus, stroke/TIA). The last INR on record was captured for all eligible patients. **RESULTS:** From 11,196,881 total patients, 58,848 patients with AF met all selection criteria. Patients at high risk of stroke ($CHADS_2 \geq 2$) **comprised 99%** (58,214) of the total patients. Among the high risk patients, 24,953 (43%) were on warfarin, 8,541 (15%) antiplatelet agents, and 24,720 (42%) neither. Of the 24,953 patients on warfarin, the last INR on record was in the suboptimal range (<2) in 13,198 (53%), above optimal range (>3) in 2,969 (12%), and in optimal range (2–3) in 8786 (35%). **CONCLUSIONS:** Analysis of a large, nationally representative EMR database suggests that the majority of AF patients at increased risk of stroke are receiving suboptimal anticoagulation.

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HEART FAILURE IN OLDER PEOPLE: A STUDY OF FACTORS THAT LEAD TO HOSPITALISATION

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OBJECTIVES: Heart failure is a frequent cause of hospitalisation among older patients. This study examines the principal factors leading to acute decompensation of heart failure leading to hospital admission and considers whether these factors are avoidable. **METHODS:** We conducted a retrospective casenote study of all patients admitted to the department of Geriatric Medicine over a 2 year period who had a principal discharge diagnosis of heart failure (ICD codes 428.0, 428.1, 428.9). Demographic and clinical data on medical history, clinical examination, radiographic findings, echocardiography, blood tests and medications was collected. Each case was evaluated to determine the factor that contributed most to heart failure decompensation. Main factors leading to hospitalisation included: volume overload if patient had all signs of hypervolaemia (pedal oedema, elevated jugular venous pressure, pulmonary crepitations and dilutional hyponatraemia), tachyarrhythmia if heart rate was >150 beats per minute, or uncontrolled hypertension if systolic blood pressure >180mmHg. **RESULTS:** The study sample included 145 patients. Mean (SD) age was 82(5) years and 74 (51%) were women. All patients were taking diuretics and 75 (51.5%) were taking angiotensin converting enzyme inhibitors. Twenty two (15.2%) were taking non steroidal anti-inflammatory drugs (NSAIDs). Risk factors for admission were identified in this rank order: volume overload 52 (35.9%) patients, chest infection 26 (17.9%) patients, combination of volume overload and chest infection 19 (13.1%) patients, myocardial infarction 17 (11.7%) patients, tachyarrhythmia (mainly atrial fibrillation) 9 (6.2%) patients, uncontrolled hypertension 10 (6.9%) and pulmonary embolus 1 (0.7%). In 11 (7.6%) patients no precipitating factor could be determined. Of the 52 patients with volume overload 12 (23.1%) were receiving NSAIDs on admission. **CONCLUSIONS:** Our findings show that factors leading to hospitalisation for heart failure may be amenable to intervention, particularly volume overload. Patient education regarding diet and medication, especially NSAIDs, and regular monitoring of blood pressure should be considered.

PCV45

DETERMINING A MODEL-DERIVED RELATIVE STROKE RISK THRESHOLD TO JUSTIFY CAROTID STENTING IN SURGICAL HIGH-RISK PATIENTS

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OBJECTIVES: To estimate the threshold of relative stroke risk needed that makes medical management inferior to protected carotid stenting in asymptomatic patients with carotid artery stenosis and substantial cardiovascular comorbidities. **METHODS:** A validated and published Monte Carlo microsimulation model created a stroke-free, two-year survival curve in monthly increments for a hypothetical medically managed arm of a recent single-arm carotid revascularization trial in patients at high surgical risk due to their cardiovascular comorbidities. Using a log rank test the actual two-year survival curve from the ACCULINK for Revascularization of Carotids in High-Risk patients (ARCHeR) trial data was compared to the medically-managed curve generated by the model. The model stroke risk equations were generated from the general population and calibrated for patients with asymptomatic carotid artery stenosis but otherwise healthy. Relative stroke risks between these patients and the surgical high-risk patients were estimated from the stroke rates of the intervention arm of a trial with more restrictive inclusion criteria (ACAS) and the strokes rates of ARCHeR. **RESULTS:** Using the best estimate of relative risk (2.02) the one- and two-year stroke rates for medical management were 0.901 and 0.851, respectively. This compares to 0.934 and 0.879 from the ARCHeR trial using protected carotid stenting. The Chi-square statistic from the log rank test of two-year survival curves was 2.26; $p = 0.13$ that medical management produces different results from carotid stenting. As long as the relative risk is greater than 2.25 the p value is <0.05. **CONCLUSIONS:** Recent carotid stenosis trials lack medically managed populations, but a model can estimate stroke-free survival and mortality data for these patients if the relative risk compared to a known population can be estimated. Since the ARCHeR trial found no ipsilateral strokes in its third year, this two-year analysis may overestimate the relative risk needed to justify carotid stenting.

PCV46

PERFLUTREN PROTEIN-TYPE A MICROSPHERES INJECTABLE SUSPENSION, USP DOES NOT INCREASE MORTALITY IN CRITICALLY ILL PATIENTS: RESULTS FROM A RETROSPECTIVE PROPENSITY-MATCHED CASE-CONTROL STUDY

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OBJECTIVES: Due to serious cardiopulmonary reactions reported immediately following administration of perflutren containing contrast agents , the United States Food and Drug Administration (FDA) required a Black Box Safety warning for this class of agents including Perflutren Protein-Type A Microspheres Injectable Suspension , USP. The FDA has requested a database analysis to compare in-hospital mortality in critically ill patients undergoing echocardiography with and without this contrast agent. This study provides results of the retrospective analysis. **METHODS:** This study utilized the largest hospital service-level database in the US. All adult patients undergoing inpatient echocardiography between January 2003 and October 2005 were identified (n = 2 ,588 ,722 of which 22 ,499 received Perflutren Protein-Type A Microspheres Injectable Suspension, USP). From the 22 ,499 contrast echocardiography patients, 2,900 had diagnoses meeting criteria for critical illness (heart failure, acute myocardial infarction, arrhythmia, respiratory failure, pulmonary embolism, emphysema , and pulmonary hypertension). To control for the differences between the contrast and non-contrast patients we used propensity score matching. Variables used in the construction of the propensity score included comorbidities , demographic factors , hospital-specific factors , level of care, and mechanical ventilation status. Patients receiving contrast echocardiography were matched to 4 controls who received non-contrast echocardiography. Conditional logistic regression was used to estimate mortality effects. **RESULTS:** There were 167 deaths in the study among critically ill patients, 38/2900 from the contrast group and 129/11600 from the control group. The contrast agent was not associated with an increase in same-day mortality (odds ratio = 1.18 (95% C.I. 0.82, 1.71); $P = 0.37$)). Prior to matching, contrast patients showed greater morbidity then non-contrast patients (Deyo-Charlson Comorbidity Score 2.45 vs. 2.25, $P < 0.0001$). After propensity-score matching these differences were significantly reduced, showing that both groups were well-balanced. **CONCLUSIONS:** There is no increase in mortality in critically ill patients undergoing echocardiography with the contrast agent compared to case-matched controls.

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THE QUANTIFICATION OF THE RELATIONSHIP BETWEEN HIGH DENSITY LIPOPROTEIN CHOLESTEROL (HDL-C) AND ALL-CAUSE MORTALITY

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OBJECTIVES: Health benefits of low-density lipoprotein cholesterol (LDL-C) at recommended levels are well known; benefits of HDL-C at recommended levels are less well documented. This study assesses and replicates the quantification of the relationship between HDL-C and all-cause mortality while aligning relative importance to LDL-C. **METHODS:** A comprehensive analyses of available epidemiological studies including HDL-C and all-cause mortality as an endpoint were included in a meta-regression. That meta-regression was independently validated with a longitudinal analysis of an Australian epidemiological database (General Practitioner Research Network) 1999–2008. While statistical association is important, demonstration that modifications via intervention to the clinical measure (HDL-C) effect change in the endpoint is more important. **RESULTS:** Twenty-two epidemiological studies in 153,798 patients met the criteria. In a survival analysis for time to death, higher HDL-C is significantly associated with longer duration to death ($P = 0.043$); 0.33 mmol/L higher HDL and 1.33 lower total/HDL cholesterol were each associated with about a third lower vascular mortality. Twenty-five placebo-controlled intervention trials yielded a regression that explained 83% of the variation in percent changes in cardiovascular events with parameter estimates associated with percent changes in